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Soman (42 µg/kg) was administered (subcutaneously) to guinea pigs (young males weighing 300-420 g). After 1 min, atropine (10 mg/kg) was administered (intramuscularly) to the animals. Simultaneously with or subsequently to (e.g., 4 min later) atropine administration, galantamine (8-10 mg/kg) was administered (intramuscularly) to the animals. Administration of 8-10 mg/kg galantamine within 5 min of administration of soman provided 100% protection. In contrast, administration of 6 mg/kg galantamine within 5 min of administration of soman only provided approximately 35% survival. In the first 24 hrs, all guinea pigs showed about a 5% weight loss; however, in the following days, the guinea pigs gained weight at the same rate as control animals that were not challenged with OPs.

All references, including publications, patent applications, and patents, cited herein are hereby incorporated by reference to the same extent as if each reference were individually and specifically indicated to be incorporated by reference and were set forth in its entirety herein.

The use of the terms “a” and “an” and “the” and similar referents in the context of describing the invention (especially in the context of the following claims) are to be construed to cover both the singular and the plural, unless otherwise indicated herein or clearly contradicted by context. Recitation of ranges of values herein are merely intended to serve as a shorthand method of referring individually to each separate value falling within the range, unless otherwise indicated herein, and each separate value is incorporated into the specification as if it were individually recited herein. All methods described herein can be performed in any suitable order unless otherwise indicated herein or otherwise clearly contradicted by context. The use of any and all examples, or exemplary language (e.g., “such as”) provided herein, is intended merely to better illuminate the invention and does not pose a limitation on the scope of the invention unless otherwise claimed. No language in the specification should be construed as indicating any non-claimed element as essential to the practice of the invention.

Preferred embodiments of this invention are described herein, including the best mode known to the inventors for carrying out the invention. It should be understood that the illustrated embodiments are exemplary only, and should not be taken as limiting the scope of the invention.

What is claimed is:

1. A method of protecting a mammal from organophosphorous (OP) poisoning, which method comprises administering to a mammal after exposure to an OP poison an OP poisoning-inhibiting amount of galantamine sufficient to diminish the detrimental effects of exposure to OP and in the absence of administration of antimuscarinic agent before exposure.

2. The method of claim 1 further comprising the step of administering to the mammal after an OP exposure that produces peripheral and central hypercholinergic signs of OP intoxication an effective amount of an antimuscarinic agent sufficient to inhibit adverse muscarinic effects of exposure to OP.

3. The method of claim 2, wherein the antimuscarinic agent is atropine.

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4. The method of claim 1 wherein the detrimental effects of OP exposure include muscarinic or nicotinic cholinergic responses.

5. The method of claim 1 wherein the detrimental effects of OP exposure are selected from the group consisting of: hyper secretion, muscle contraction, respiratory difficulties, convulsion, and behavioral abnormalities, and combinations thereof.

6. The method of claim 1 wherein the detrimental effects of OP exposure comprises loss of neuronal viability and protecting a mammal from OP poisoning includes administering galantamine in an amount sufficient to preserve or restore neuronal structures.

7. The method of claim 1 wherein galantamine is galantamine hydrobromide.

8. The method of claim 1 wherein galantamine is administered in an OP poisoning-inhibiting amount determined in accordance with dosage range-finding techniques.

9. The method of claim 1 wherein galantamine is administered in a clinical setting or in the field in an OP-poisoning inhibiting amount, the amount determined by administering an initial dose and then incrementally altering the initial dose to achieve an optimum effect under the circumstances.

10. The method of claim 1 wherein galantamine is administered in an amount effective to arrest OP-induced toxicity, the effective amount determined as establishing galantamine-induced cholinesterase inhibition in the brain of from a negligible amount to about 20%.

11. The method of claim 10 wherein the amount of cholinesterase inhibition established in the brain ranges from less than 1% to about 20%.

12. The method of claim 1 wherein galantamine is administered orally or intramuscularly.

13. A method of protecting a mammal from loss of neuronal viability by exposure to organophosphorous (OP) poison, which method comprises administering to a mammal after exposure to an OP poison, and in the absence of administering antimuscarinic agent before exposure, an amount of galantamine sufficient to preserve neuronal structures.

14. The method of claim 13 wherein galantamine is administered in an amount sufficient to rescue nicotinic receptors from desensitization or to protect against desensitization.

15. The method of claim 13 wherein galantamine is administered in an amount sufficient to preserve neuronal structures, the amount determined by administering an initial dose and then incrementally altering the initial dose to achieve an optimum effect on behavioral abnormalities under the circumstances.

16. The method of claim 13 wherein galantamine is galantamine hydrobromide.

17. A method for rescuing nicotinic receptors from and restoring or preserving neuronal viability after OP exposure, the method comprising the step of administering to a mammal following OP exposure, and in the absence of administering antimuscarinic agent prior to exposure, an amount of galantamine hydrobromide sufficient to establish galantamine-induced cholinesterase inhibition in the brain of from a negligible amount to about 20%.

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